

09/978,146

PALM INTRANET

Day : Wednesday

Date: 12/8/2004

Time: 14:34:06

**Inventor Name Search Result**

Your Search was:

Last Name = MELMED

First Name = SHLOMO

Application#	Patent#	Status	Date Filed	Title	Invent Name
<u>60370912</u>	Not Issued	159	04/08/2002	OVER-EXPRESSION OF THE MAMMALIAN SECURIN, PTTG, DISRUPTS MITOSIS AND LEADS TO ANEUPLOIDY	MELM SHLO
<u>60045241</u>	Not Issued	159	05/01/1997	METHOD OF TREATING HYPERPROLACTINEMIA AND PROLACTINOMAS	MELM SHLO
<u>60031338</u>	Not Issued	159	11/21/1996	NUCLEIC ACID ENCODING A FAMILY OF PITUITARY-TUMOR-SPECIFIC-GENES, AND PRODUCTS RELATED THERETO	MELM SHLO
<u>10334385</u>	Not Issued	061	12/31/2002	SUPPRESSOR OF CYTOKINE SIGNALING (SOCS)-3 PROMOTER AND METHODS FOR ITS USE IN GENETIC THERAPY IN HUMANS	MELM SHLO
<u>10284126</u>	Not Issued	030	10/29/2002	POLYNUCLEOTIDES ENCODING MOUSE PITUITARY TUMOR TRANSFORMING GENE (PTTG) CARBOXY-TERMINAL PEPTIDES AND METHODS OF USE THEREOF TO INHIBIT NEOPLASTIC CELLULAR PROLIFERATION AND/OR TRANSFORMATION	MELM SHLO
<u>10283874</u>	Not Issued	030	10/29/2002	POLYNUCLEOTIDES ENCODING RAT PITUITARY TUMOR TRANSFORMING GENE (PTTG) CARBOXY-TERMINAL PEPTIDES AND METHODS OF USE THEREOF TO INHIBIT NEOPLASTIC CELLULAR PROLIFERATION AND/OR TRANSFORMATION	MELM SHLO
<u>10283797</u>	Not Issued	041	10/29/2002	NON-HUMAN MAMMALS COMPRISING CELLS EXPRESSING VECTOR-BORNE MOUSE PTTG CARBOXY-TERMINAL-RELATED DNA	MELM SHLO
<u>10283771</u>	Not Issued	041	10/29/2002	NON-HUMAN MAMMALS COMPRISING CELLS EXPRESSING VECTOR-BORNE RAT PTTG CARBOXY-TERMINAL-RELATED DNA	MELM SHLO
<u>10264372</u>	Not Issued	041	10/04/2002	TRANSGENIC CELLS TRANSFECTED WITH PITUITARY TUMOR TRANSFORMING GENE (PTTG)) EXPRESSION VECTORS AND USES THEREFOR	MELM SHLO

<u>10262264</u>	Not Issued	030	09/30/2002	OLIGONUCLEOTIDES ANTISENSE TO MOUSE PITUITARY TUMOR TRANSFORMING GENE CARBOXY-TERMINAL (PTTG-C) AND METHODS OF USE THEREOF TO INHIBIT NEOPLASTIC CELLULAR PROLIFERATION AND/OR TRANSFORMATION	MELN SHLO
<u>10262258</u>	Not Issued	030	09/30/2002	OLIGONUCLEOTIDES ANTISENSE TO RAT PITUITARY TUMOR TRANSFORMING GENE CARBOXY-TERMINAL (PTTG-C) AND METHODS OF USE THEREOF TO INHIBIT NEOPLASTIC CELLULAR PROLIFERATION AND/OR TRANSFORMATION	MELN SHLO
<u>10262252</u>	Not Issued	071	09/30/2002	ANTIBODIES AGAINST MOUSE PITUITARY TUMOR TRANSFORMING GENE CARBOXY-TERMINAL (PTTG-C) PEPTIDES	MELN SHLO
<u>10261821</u>	Not Issued	095	09/30/2002	ANTIBODIES AGAINST RAT PITUITARY TUMOR TRANSFORMING GENE CARBOXY-TERMINAL (PTTG-C) PEPTIDES	MELN SHLO
<u>10261787</u>	Not Issued	041	09/30/2002	RAT PITUITARY TUMOR TRANSFORMING GENE (PTTG) CARBOXY-TERMINAL PEPTIDES AND METHODS OF USE THEREOF TO INHIBIT NEOPLASTIC CELLULAR PROLIFERATION AND/OR TRANSFORMATION	MELN SHLO
<u>10261717</u>	Not Issued	041	09/30/2002	MOUSE PITUITARY TUMOR TRANSFORMING GENE (PTTG) CARBOXY-TERMINAL PEPTIDES AND METHODS OF USE THEREOF TO INHIBIT NEOPLASTIC CELLULAR PROLIFERATION AND/OR TRANSFORMATION	MELN SHLO
<u>10252309</u>	Not Issued	030	09/23/2002	LIVE CELL METHOD FOR OBSERVING CELLULAR PROCESSES	MELN SHLO
<u>10183140</u>	Not Issued	161	06/25/2002	PITUITARY-TUMOR-TRANSFORMING-GENES, AND RELATED PRODUCTS	MELN SHLO
<u>10176812</u>	Not Issued	061	06/21/2002	TRANSGENIC NON-HUMAN MAMMALS CARRYING HUMAN PITUITARY TUMOR TRANSFORMING GENE (PTTG) SEQUENCES	MELN SHLO
<u>10176549</u>	Not Issued	061	06/21/2002	TRANSGENIC NON-HUMAN MAMMALS CARRYING RAT PITUITARY TUMOR TRANSFORMING GENE (PTTG) SEQUENCES	MELN SHLO
<u>10163277</u>	Not Issued	041	06/04/2002	PITUITARY-TUMOR-TRANSFORMING-GENES, AND RELATED PRODUCTS	MELN SHLO
<u>10163053</u>	6673823	150	06/04/2002	USE OF PEROXISOME PROLIFERATOR ACTIVATED RECEPTOR (PPAR)-GAMMA LIGANDS AS A TREATMENT FOR PITUITARY TUMORS AND ASSOCIATED CONDITIONS, SUCH AS CUSHING'S SYNDROME	MELN SHLO

<u>10136224</u>	Not Issued	061	04/29/2002	TRANSGENIC EXPRESSION FROM A SOCS-3 PROMOTER IN VERTEBRATE CELLS	MELN SHLO
<u>10136098</u>	Not Issued	041	04/29/2002	OLIGONUCLEOTIDES ANTISENSE TO PITUITARY TUMOR TRANSFORMING GENE CARBOXY-TERMINAL (PTTG-C) AND METHODS OF USE THEREOF TO INHIBIT NEOPLASTIC CELLULAR PROLIFERATION AND/OR TRANSFORMATION	MELN SHLO
<u>10136082</u>	Not Issued	094	04/29/2002	PITUITARY TUMOR TRANSFORMING GENE (PTTG) CARBOXY-TERMINAL PEPTIDES AND METHODS OF USE THEREOF TO INHIBIT NEOPLASTIC CELLULAR PROLIFERATION AND/OR TRANSFORMATION	MELN SHLO
<u>10136056</u>	Not Issued	061	04/29/2002	NON-HUMAN MAMMALS COMPRISING CELLS EXPRESSING VECTOR-BORNE PTTG CARBOXY-TERMINAL-RELATED DNA	MELN SHLO
<u>10135671</u>	Not Issued	041	04/29/2002	ANTIBODIES AGAINST PITUITARY TUMOR TRANSFORMING GENE CARBOXY-TERMINAL (PTTG-C) PEPTIDES	MELN SHLO
<u>10124905</u>	Not Issued	041	04/17/2002	ANTI-INFLAMMATORY THERAPIES USING CYTOKINE SIGNALING REGULATED BY A SOCS-3 PROMOTER	MELN SHLO
<u>09978146</u>	Not Issued	080	10/15/2001	PTTG KNOCKOUT RODENT AS A MODEL TO STUDY MECHANISMS FOR VARIOUS PHYSIOLOGICAL PHENOMENA, INCLUDING DIABETES	MELN SHLO
<u>09949476</u>	6750327	150	09/07/2001	COMPOSITIONS AND METHOD FOR DETERMINING THE PRESENCE OF HUMAN PTTG PEPTIDE IN A SAMPLE	MELN SHLO
<u>09949272</u>	Not Issued	094	09/07/2001	HUMAN PTTG POLYPEPTIDE AND METHOD FOR PRODUCING IT	MELN SHLO
<u>09949271</u>	6723519	150	09/07/2001	COMPOSITIONS AND METHOD FOR DETERMINING THE PRESENCE OF RAT PTTG PEPTIDE IN A SAMPLE	MELN SHLO
<u>09949270</u>	Not Issued	094	09/07/2001	RAT PTTG POLYPEPTIDE AND METHOD FOR PRODUCING IT	MELN SHLO
<u>09854326</u>	Not Issued	094	05/11/2001	METHOD OF REGULATING BIOLOGICAL ACTIVITY OF PITUITARY TUMOR TRANSFORMING GENE (PTTG)1 USING PTTG2	MELN SHLO
<u>09777422</u>	Not Issued	161	02/05/2001	METHODS OF MODULATING ANGIOGENESIS BY REGULATING THE EXPRESSION OF PITUITARY TUMOR TRANSFORMING GENE (PTTG)	MELN SHLO
<u>09730469</u>	Not Issued	161	12/04/2000	METHODS OF USING PITUITARY TUMOR TRANSFORMING GENE (PTTG) CARBOXY-TERMINAL PEPTIDES TO INHIBIT	MELN SHLO

				NEOPLASTIC CELLULAR PROLIFERATION AND/OR TRANSFORMATION OF BREAST AND OVARIAN CELLS	
<u>09687911</u>	Not Issued	061	10/13/2000	MODULATING ACTIVATION OF LYMPHOCYTES AND SCREENING POTENTIAL IMMUNOMODULATING AGENTS BY TARGETING PITUITARY TUMOR TRANSFORMING GENE (PTTG) EXPRESSION AND/OR FUNCTION	MELN SHLO
<u>09569956</u>	Not Issued	094	05/12/2000	PITUITARY TUMOR TRANSFORMING GENE (PTTG) CARBOXY-TERMINAL PEPTIDES AND METHODS OF USE THEREOF TO INHIBIT NEOPLASTIC CELLULAR PROLIFERATION AND/OR TRANSFORMATION	MELN SHLO
<u>09327138</u>	<u>6541244</u>	150	06/07/1999	SUPPRESSOR OF CYTOKINE SIGNALING (SOCS)-3 PROMOTER AND METHODS FOR ITS USE IN GENETIC THERAPY IN HUMANS	MELN SHLO
<u>08894251</u>	<u>6455305</u>	150	07/23/1999	PITUITARY-TUMOR-TRANSFORMING-GENES, AND RELATED PRODUCTS	MELN SHLO
<u>08852221</u>	<u>5972893</u>	150	05/06/1997	METHOD OF TREATING HYPERPROLACTINEMIA AND PROLACTINOMA	MELN SHLO
<u>08848787</u>	Not Issued	169	05/01/1997	METHOD OF TREATING HYPERPORLACTINEMIA AND PROLACTINOMAS	MELN SHLO
<u>08647401</u>	<u>5824838</u>	150	05/09/1996	TRANSGENIC MOUSE MODEL FOR PITUITARY DISORDERS ASSOCIATED WITH LIF OVEREXPRESSION AND/OR GH UNDEREXPRESSION, AND ITS USE FOR TESTING THERAPEUTIC DRUGS FOR THE CONDITIONS	MELN SHLO
<u>08465232</u>	Not Issued	164	06/05/1995	VARIANT INSULIN-LIKE GROWTH FACTOR I RECEPTOR SUBUNITS AND METHODS FOR USE THEREOF	MELN SHLO
<u>08460787</u>	Not Issued	164	06/05/1995	INHIBITION OF RECEPTOR FUNCTION WITH USE OF VARIANT INSULIN-LIKE GROWTH FACTOR I RECEPTOR SUBUNITS	MELN SHLO
<u>08249687</u>	<u>5942412</u>	250	05/26/1994	POLYNUCLEIC ACID ENCODING VARIANT INSULIN-LIKE GROWTH FACTOR I RECEPTOR	MELN SHLO
<u>08044540</u>	Not Issued	166	04/06/1993	VARIANT INSULIN-LIKE GROWTH FACTOR I RECEPTOR SUBUNITS AND METHODS FOR USE THEREOF	MELN SHLO

Inventor Search Completed: No Records to Display.

**Search Another:  
Inventor**

**Last Name**

melmed

**First Name**

shlomo

Search

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09/9/78,146

=> d his

(FILE 'HOME' ENTERED AT 14:45:18 ON 08 DEC 2004)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 14:45:31 ON 08 DEC 2004

L1 1137 S PTTG OR PTSG OR SECURIN  
L2 76705 S (NULL(W)MUTANT OR KNOCKOUT) (5A) (MOUSE OR MICE OR RAT OR RODEN  
L3 1 S L1(S)L2  
L4 4 S L1 AND L2  
L5 4 DUP REM L4 (0 DUPLICATES REMOVED)

=> d bib ab 13

L3 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2004 ACS on STN  
AN 2003:396993 CAPLUS  
DN 138:397254  
TI **PTTG knockout rodent** as a model to study  
mechanisms for various physiological phenomena, including diabetes  
IN Wang, Zhiyong; Melmed, Shlomo  
PA Cedars-Sinai Medical Center, USA  
SO PCT Int. Appl., 50 pp.  
CODEN: PIXXD2

DT Patent  
LA English  
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2003042356	A2	20030522	WO 2002-US30845	20020927
	WO 2003042356	A3	20031016		
	W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW			
	RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
	US 2003106080	A1	20030605	US 2001-978146	20011015
	EP 1435775	A2	20040714	EP 2002-773633	20020927
	R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK			
PRAI	US 2001-978146	A	20011015		
	WO 2002-US30845	W	20020927		

AB The present invention discloses a **null mutant** (or **knockout**) **rodent** comprising in its germ cells an artificially induced **PTTG** null mutation. In some embodiments, the null mutant rodent can be generated by way of homologous recombination in an embryonic stem cell or germ cell. The inventive null mutant rodent can be used to study mammalian physiol. at the cellular, tissue, and/or organismal level with respect to various phenotypes, including hyperglycemia, hypoinsulinemia, hypoleptinemia, diabetes, chromosomal aneuploidy, premature centromere division, chromosomal damage, aberrant mitotic cellular division, thrombocytopenia, thymic hyperplasia, splenic hypoplasia, testicular hypoplasia, and female subfertility. Also disclosed is an animal model for diabetes, a somatic or germ cell obtained from the null mutant rodent and a cell line derived from a cell obtained from the null mutant rodent.

=> d au ti so pi ab 1-4 15

L5 ANSWER 1 OF 4 MEDLINE on STN  
 AU Dai Wei; Wang Qi; Liu Tongyi; Swamy Malisetty; Fang Yuqiang; Xie Suqing;  
 Mahmood Radma; Yang Yang-Ming; Xu Ming; Rao Chinthalapally V  
 TI Slippage of mitotic arrest and enhanced tumor development in mice with  
 BubR1 haploinsufficiency.  
 SO Cancer research, (2004 Jan 15) 64 (2) 440-5.  
 Journal code: 2984705R. ISSN: 0008-5472.  
 AB A compromised spindle checkpoint is thought to play a key role in genetic  
 instability that predisposes cells to malignant transformation. Loss of  
 function mutations of BubR1, an important component of the spindle  
 checkpoint, have been detected in human cancers. Here we show that  
 BubR1(+/-) mouse embryonic fibroblasts are defective in spindle checkpoint  
 activation, contain a significantly reduced amount of **securin**  
 and Cdc20, and exhibit a greater level of micronuclei than do wild-type  
 cells. RNA interference-mediated down-regulation of BubR1 also greatly  
 reduced **securin** level. Moreover, compared with wild-type  
 littermates, BubR1(+/-) mice rapidly develop lung as well as intestinal  
 adenocarcinomas in response to challenge with carcinogen. BubR1 is thus  
 essential for spindle checkpoint activation and tumor suppression.

L5 ANSWER 2 OF 4 MEDLINE on STN  
 AU Wirth Karin G; Ricci Romeo; Gimenez-Abian Juan F; Taghybeeglu Shahryar;  
 Kudo Nobuaki R; Jochum Wolfram; Vasseur-Cognet Mireille; Nasmyth Kim  
 TI Loss of the anaphase-promoting complex in quiescent cells causes  
 unscheduled hepatocyte proliferation.  
 SO Genes & development, (2004 Jan 1) 18 (1) 88-98.  
 Journal code: 8711660. ISSN: 0890-9369.  
 AB The anaphase-promoting complex or cyclosome (APC/C) is an ubiquitin  
 protein ligase that together with Cdc20 and Cdh1 targets mitotic proteins  
 for degradation by the proteasome. APC-Cdc20 activity during mitosis  
 triggers anaphase by destroying **securin** and cyclins. APC-Cdh1  
 promotes degradation of cyclins and other proteins during G(1). We show  
 that loss of APC/C during embryogenesis is early lethal before embryonic  
 day E6.5 (E6.5). To investigate the role of APC/C in quiescent cells, we  
 conditionally inactivated the subunit Apc2 in mice. Deletion of Apc2 in  
 quiescent hepatocytes caused re-entry into the cell cycle and arrest in  
 metaphase, resulting in liver failure. Re-entry into the cell cycle  
 either occurred without any proliferative stimulus or could be easily  
 induced. We demonstrate that the APC has an additional function to  
 prevent hepatocytes from unscheduled re-entry into the cell cycle.

L5 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN  
 IN Wang, Zhiyong; Melmed, Shlomo  
 TI **PTTG knockout rodent** as a model to study  
 mechanisms for various physiological phenomena, including diabetes  
 SO PCT Int. Appl., 50 pp.  
 CODEN: PIXXD2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003042356	A2	20030522	WO 2002-US30845	20020927
WO 2003042356	A3	20031016		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG US 2003106080 A1 20030605 US 2001-978146 20011015 EP 1435775 A2 20040714 EP 2002-773633 20020927 R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,				

IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK  
AB The present invention discloses a **null mutant** (or **knockout**) **rodent** comprising in its germ cells an artificially induced **PTTG** null mutation. In some embodiments, the **null mutant rodent** can be generated by way of homologous recombination in an embryonic stem cell or germ cell. The inventive **null mutant rodent** can be used to study mammalian physiol. at the cellular, tissue, and/or organismal level with respect to various phenotypes, including hyperglycemia, hypoinsulinemia, hypoleptinemia, diabetes, chromosomal aneuploidy, premature centromere division, chromosomal damage, aberrant mitotic cellular division, thrombocytopenia, thymic hyperplasia, splenic hypoplasia, testicular hypoplasia, and female subfertility. Also disclosed is an animal model for diabetes, a somatic or germ cell obtained from the **null mutant rodent** and a cell line derived from a cell obtained from the **null mutant rodent**.

L5 ANSWER 4 OF 4 MEDLINE on STN  
AU Wang Zhiyong; Moro Enrico; Kovacs Kalman; Yu Run; Melmed Shlomo  
TI Pituitary tumor transforming gene-null male mice exhibit impaired pancreatic beta cell proliferation and diabetes.  
SO Proceedings of the National Academy of Sciences of the United States of America, (2003 Mar 18) 100 (6) 3428-32.  
Journal code: 7505876. ISSN: 0027-8424.  
AB The mammalian **securin**, pituitary tumor transforming gene (**PTTG**), regulates sister chromatid separation during mitosis. Mice or cell lines deficient in **PTTG** expression, however, are surprisingly viable. Here we show that **PTTG** disruption in mice (**PTTG**<sup>-/-</sup>) severely impairs glucose homeostasis leading to diabetes during late adulthood, especially in males associated with nonautoimmune insulinopenia and reversed alphabeta cell ratio. Islet beta cell mass in **PTTG**<sup>-/-</sup> mice was already diminished before development of frank diabetes and only increased minimally during growth. BrdUrd incorporation of islet cells in **PTTG**-null mice was approximately 65% lower (P < 0.005) than in the WT pancreas, whereas apoptosis rates were similar. **PTTG**<sup>-/-</sup> beta cells had pleiotropic nuclei, suggesting defects in cell division. The results indicated that **securin** is indispensable for normal pancreatic beta cell proliferation.

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## Refine Search

### Search Results -

Terms	Documents
L1 with L2	1

Database:

US Pre-Grant Publication Full-Text Database  
 US Patents Full-Text Database  
 US OCR Full-Text Database  
 EPO Abstracts Database  
 JPO Abstracts Database  
 Derwent World Patents Index  
 IBM Technical Disclosure Bulletins

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### Search History

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<u>Set</u> <u>Name</u> side by side	<u>Query</u>	<u>Hit</u> <u>Count</u>	<u>Set</u> <u>Name</u> result set
	DB=PGPB,USPT; PLUR=YES; OP=AND		
<u>L3</u>	l1 with L2	1	<u>L3</u>
<u>L2</u>	(null adj mutant or knockout) near5 (mouse or mice or rodent or rat or mammal or animal)	7721	<u>L2</u>
<u>L1</u>	pttg or ptsg or securin	109	<u>L1</u>

END OF SEARCH HISTORY

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- ☐ 1. 20030106080. 15 Oct 01. 05 Jun 03. PTTG knockout rodent as a model to study mechanisms for various physiological phenomena, including diabetes. Melmed, Shlomo, et al. 800/14; 435/353 435/354 800/18 A01K067/027 C12N005/06.

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Terms	Documents
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